Adams (S.S.)

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Lecturer on Diseases of Children, Medical Department University of Georgetown, D. C.

Reprinted from the AMERICAN JOURNAL OF OBSTETRICS AND DISEASES OF WOMEN AND CHILDREN, *Vol. XVII., No.* 8.

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Though fully aware that diphtheria has been exhaustively discussed by many of our best authors, still the reintroduction of the subject may be pardonable when we have in view the recent reports of sudden deaths occurring during the decline of the disease and the backwardness of the reporters in venturing an opinion as to the probable cause of this sudden dissolution.

In the case upon which this paper is based there are many interesting points which I will endeavor to explain. By comparing this with other cases I hope to be able to show that sudden death is not invariably due to paralysis of the heart.

On the 23d of December, 1883, my attention was called to Chester A., a bright bouncing boy of sixteen months, well-developed, with good constitution, twelve teeth, and a fontanele normal at the age. His mother said he occasionally put his hands to his head, and grunted as if in pain. As he was then running merrily about the room and into the kitchen, and as he was accustomed to imitate the actions of the elder members of the family, I told them that apparently he was well, and advised them to let him alone.

At this time the mother, a healthy lady, complained of feeling languid, but went about and ate as usual, and did not ask for advice.

At 4 A.M., December 24th, the father came for me in great haste, saying that his baby was dying of convulsions. I hastened to the house on De Sales street, and learned that a homeopathic physician had been there, had pronounced it a case of difficult dentition, and left some *aconite* in a tumblerful of water.

The child was now sleeping very quietly; skin hot and dry; respiration jerky; fontanelle depressed; pulse rapid, with good tension; tongue fair; no muscular twitchings; gums neither red nor swollen.

I obtained the following history: The child had gone to bed feeling bright, and the members of the family had been called in to see his antics. In the early hours of morning he began to be restless, fretted, and seemed agitated, but his parents thought it mischievousness, and scolded him; he would keep quiet for a while, but very soon would repeat the movements. For a time all slept, when the mother was suddenly awakened by the peculiar noises he made, felt him to be rigid, and sprang from the bed, running into her mother's room to the light, to find that her babe was in a severe convulsion. This attack was described as lasting ten minutes. Upon inquiry I learned that the diet of the previous day had been of the simplest, consisting of bread, potatoes, and milk; he had not had any sweetmeats or fruit. Knowing that the little fellow was accustomed to spend most of his time running about the kitchen, I directed my attention to the probability of his having obtained some indigestible substance, and his nurse, who was greatly alarmed at his condition, admitted that the day before he did get some potato rinds, but that he had only eaten a little when she discovered it.

This gave me a clue, a I supposed, and I ventured the opinion that the convulsion was due either to some indigestible material in the alimentary tract or to the development of some acute disease, and that I favored the former view.

The child had a natural stool the day before, but my first aim was to rid the alimentary canal of any offending material. For this purpose I gave an enema of warm water, which was retained ten or fifteen minutes; ordered calomel gr. i. every hour until two were taken. To quiet the nervous system he was given the potassium bromide gr. iij. every hour.

Anticipating further trouble, I lay on the bed in a distant part of the room. He slept at intervals, and when awake manifested no signs of returning convulsions. At 8 A.M., he was lying on the lounge resting so quietly that his grandmother left the room for a few moments. I was awakened by a peculiar cry that seems to be characteristic, and going to him found that he was then in an eclamptic attack, which could by no means be called severe, and which lasted about two minutes. I now gave another enema which soon came away without any fecal matter. I now directed that if by 9 o'clock he had not a stool, to give two more of the calomel powders as before.

As I was about leaving the house the mother asked me to look at her throat, as it felt sore. Her skin was hot and dry, pulse bounding. Upon the right tonsil was a dirty patch about the size of a pea. I ordered her to bed, and refused to state the nature of the patch, but favored the view that it was aphthous, saying that at my next visit I would tell her what it was.

This unexpected discovery caused me to return to the baby and examine his throat. His tonsils were slightly enlarged and intensely hyperemic, but there was not the slightest trace of a membrane.

I visited the patients at 2 P.M., and learned that about 10 o'clock, after taking the two powders, the baby had a copious stool consisting of fecal matter mixed with potato rinds, cranberry seed, and undigested particles of orange and potato. At 11 A.M. he had a convulsion, which was described as being very slight. He did not seem very much exhausted; skin slightly moist; tongue dry; fontanelle depressed; pulse frequent, but good. Again examined his throat, but saw no evidences of membrane. Continued the potassium bromide; milk diet.

I now directed attention to the mother, and found a quick, bounding pulse, hot, dry skin, tendency to sleep, tongue covered with a dirty brown fur, and two-thirds of the right tonsil covered by a thick, dirty membrane. I could now pronounce the case one of diphtheria. I enjoined absolute rest in bed, liquid diet, and the proper local applications.

The two patients, with the child's father and grandmother, were now isolated, and every precaution taken to prevent the spread of the disease among the family, including three children, the eldest not yet five years of age.

At 7 P. M. I saw the patients. The baby had gotten along very well after my last visit until 5 o'clock, when he had the fourth and last convulsion, which was described as being almost as severe as the first. He had a hot, dry skin, very restless, pulse rapid and full, respiration quick; had not taken any milk, and had slept at intervals. Upon examining his throat I found the right tonsil nearly covered with a membrane; his pulse 150, and a temperature in the axilla of 104.5°. I now pronounced this a case of diphtheria. From the age of the patient, the accompanying convulsions, and the fact that several years ago a brother had died on the second day of diphtheria, I felt justified in pointing out the extreme gravity of the case, and expressed a desire to have Dr. Samuel C. Busey called in consultation, which was allowed.

Dr. Busey soon met me. He thoroughly examined the mother and child, confirmed the diagnosis and agreed with me in making an unfavorable prognosis.

As the further history of the mother's case is not germane to the subject, and as later on I will explain my motive for introducing so much of it, I will drop it to complete the progress of the child's case.

We thought the convulsions were under the control of the bromide, but still deemed it advisable to continue it during the night with instructions to increase the dose should they recur. To reduce the temperature sodium salicylate, gr. iij. every two hours, was ordered, though we were apprehensive of its depressing effect upon the heart. Throat to be sprayed every two hours with the

hand-ball atomizer, containing a mixture of carbolic acid, chlorate of potassium, tincture of the ferric chloride, glycerin, and water. As he had not taken any nourishment during the day we insisted upon the milk and lime-water being pushed. A pan of lime and water, in the room near where the child lay, was kept boiling during the entire illness. I remained with the patient until midnight, and, after instructing those in charge how to use the atomizer, left him resting quietly.

December 25th, 8 A.M. P. 144, R. 28, temp. 99.4°. Has had no convulsions, slept very well; surface cool and moist, one stool; urinated freely three times; bright, and expression better; has taken half a pint of milk.

10 A.M. Dr. Busey in consultation, P. 138, R. 32, temp. 100.2°. As the fever was lower, the heart depressed, and there being no evidences of recurring convulsions, it was determined to stop the sodium and potassium, and give a grain of the hydrochlorate of quinia every four hours. The character of the pulse and the depression of the fontanelle indicated the necessity of stimulants, so fifteen drops of whiskey every two hours were ordered. The membrane was extending over the uvula and left tonsil, but there was no difficulty in breathing. He fought so against the spray, and being afraid the tube would injure him, it was directed to be used every four hours.

2 P.M. P. 146, R. 40, T. 101.4°. No material change.

8 P.M. Dr. Busey in consultation, P. 165, R. 36, T. 103.2°. No stool; but little sleep; uneasy and fretful; has taken a pint of milk; seems to have no pain in swallowing; urinated freely; tonsils and uvula covered with membrane. Increase whiskey to twenty drops. Continue the other treatment.

December 26th, 8 A.M. P. 145, R. 36, T. 100.2°. Slept a good deal, but not quietly; occasionally awoke screaming; fontanelle depressed; pulse tension poor; skin comfortable; one free stool; urinates freely. One pint of milk taken.

10 A.M. Dr. Busey in consultation. Throat looks about the same. Discontinue quinine. Whiskey thirty drops.

2 P.M. P. 140, R. 24, temp. 102°.

8 P.M. Dr. Busey in consultation. P. 140, R. 30, T. 101.6°. Has passed a restless day, no stool; not a pint of milk taken.

December 27th, 8 A.M. P. 152, R. 32, T. 100°. Restless and fretful during the night; no stool; right tonsil clean, small patches on left and uvula. Took a quart of milk.

10 A.M. Dr. Busey in consultation. Slight improvement.

2 P.M. P. 142, tension good, R. 24, temp. 101.4°.

8 P.M. Dr. Busey in consultation. P. 160, tension bad; R. 30, T. 101.8°. Has been bright and cheerful all day; played with his toys for the first time; one large pasty stool; no membrane perceptible; a few large moist râles heard over both lungs. Has taken a pint of milk. Same treatment and one grain of the quinine every four hours.

December 28th, 8 A.M. P. 140, R. 22, temp. 100.6°. Passed

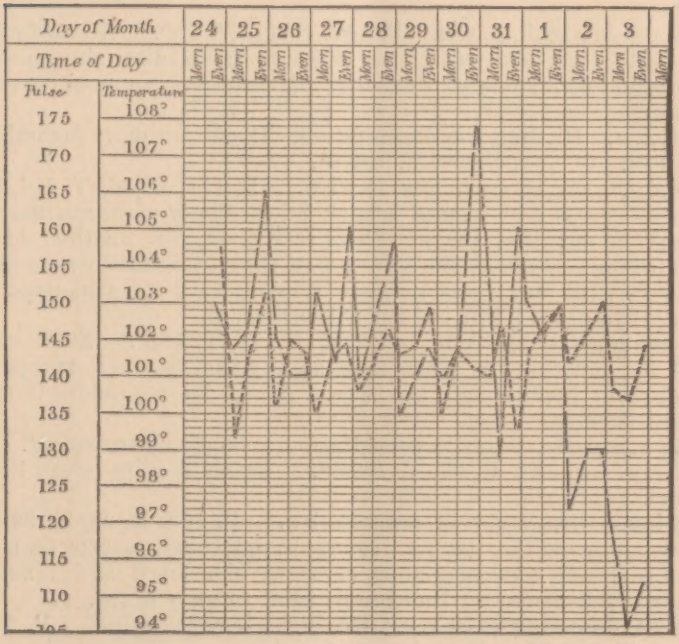
a very restless night; nose seemed to be stopped, which awakened him at short intervals; fretful; one good stool; membrane has reappeared on right tonsil; râles increasing, a few crepitant. Oil silk jacket, and ammonium carbonate, gr. i. every four hours ordered.

10 A.M. Dr. Busey in consultation. No change.

2 P.M. P. 148, R. 33, temp. 101.4°.

8 P.M. Dr. Busey in consultation. P. 158, R. 35, T. 102.2°. Has passed a very comfortable day, breathing through nose easier; has taken sufficient nourishment. Continue treatment.

December 29th, 8 A.M. P. 135, R. 24, T. 101.6°. Slept very



well during the night; membrane has reappeared on the left tonsil; very little nourishment during the night; a few râles over right lung.

10 A.M. Dr. Busey in consultation. Condition as above described; no change in treatment.

2 P.M. P. 140, R. 28, T. 101.8°. Has been sitting on his mother's bed playing with toys.

(At this visit my attention was called to Mrs. R., grandmother of the child. She had flushed face, coated tongue, hot dry skin, pulse 120, R. 27, T. 101.8°, pain in back of neck, and sore throat; a large patch of membrane on right tonsil. She was put

to bed and a professional nurse obtained. It may be of interest to note that up to this time this lady had almost continuously held the baby in her arms, and in spite of my protestations, persisted in fondling and kissing him, so that there is nothing strange about the poison infecting her. She was put under systematic treatment.)

8 P.M. Dr. Busey in consultation. P. 144, R. 29, T. 102.8°. All in all he has had a very comfortable day; one stool; urinated frequently; a few sonorous râles; has taken large quantities of beef-tea and milk. As the temperature did not yield to the quinine it was stopped. The little fellow fought so hard against having his throat inspected, as each attempt made it bleed, and as he was breathing easily, it was deemed prudent to let him alone.

December 30th, 8 A.M. P. 140, R. 21, T. 100. A comfortable night; breathed easily; one large stool; no râles; skin moist; expression good; one pint of milk. Discontinue ammonium carbonate.

10 A.M. Dr. Busey in consultation. Condition more favorable.

2 P.M. P. 144, R. 28, T. 101.6°.

8 P.M. Dr. Busey in consultation. P. 174, R. 36, T. 101.2°. A comfortable day, seemed bright; takes plenty of nourishment. It will be seen that the pulse was very rapid, but we thought it was principally due to the agitation of the patient at our presence. However, the nurse was instructed to increase the stimulant to thirty-five drops if the pulse continued rapid.

December 31st, 8 A.M. P. 160, R. 28, T. 101°. Slept well, and his breathing is easier than at any time before; two large stools; but little cough; no râles. Asked for nourishment and took it freely.

10 A.M. Dr. Busey in consultation. Continue treatment.

2 P.M. P. 124. R. 32, T. 102.4°. Renew quinine every three hours.

8 P.M. Dr. Busey in consultation. P. 160, R. 29, T. 99.6°. Rested during the day; three stools, no cough; seems to have pain in head; cervical glands of right side hard and tender; takes sufficient food. Increase whiskey to forty drops.

1884, January 1st, 10 A.M. Dr. Busey in consultation. P. 150, R. 40, T. 101.8°. Passed a quiet night; no cough; pain in neck seems to be less, though the swelling has increased; one stool, sufficient nourishment.

2 P.M. P. 130, R. 56, temp. 102.2°.

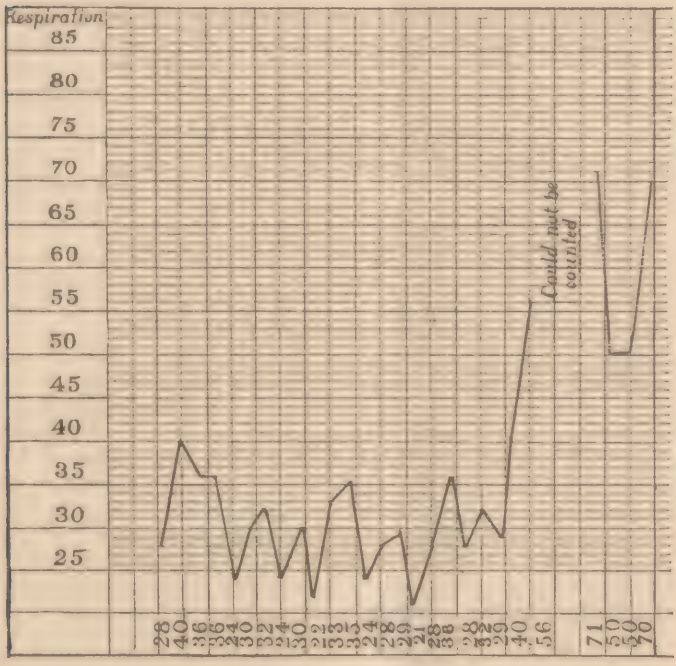
8 P.M. Dr. Busey in consultation. P. 150, R. —, temp. 102.8°. Was restless and fretful all the afternoon, and vomited once, since which his condition has been better than at any time during the day.

January 2d, 10 A.M. P. 122, R. —, 101.4°. Vomited five times during the night; slept but little; pain in head; two good stools; awakes screaming as if frightened by dreams. Owing to the irritability of the stomach the quinine was stopped. Hardly any nourishment during the night.

2 P.M. P. 130, R. —, T. 102.2°.

5 P.M. P. 130, R. 71, T. 103°. Has passed a very uncomfortable day; stomach has rejected nearly everything. Stimulants and food only.

January 3d, 10 A.M. Dr. Busey in consultation. P. 120, R. 50, T. 100.6°. Was sent for to see the patient at 9 o'clock last night. He had vomited three times since my visit at 5. Stimulants and food were stopped to give the stomach a rest. Ordered one-quarter drop of creasote in a drachm of lime-water every hour until easier. Visited him again at midnight. Had taken two doses of the medicine, had not vomited, and was then sleeping



quietly. The stomach to rest until early morning, when he was to have milk and lime-water in teaspoonful doses every ten or fifteen minutes. This morning he seems greatly improved; pulse tension remarkably good; has retained large quantities of milk; cervical glands increasing and very tender; no lung complication to account for the rapid respiration, nor any obstruction in the upper air-passages. His condition is very encouraging, though we will not venture a prognosis.

2 P.M. P. 106, R. 50, T. 100.4°. Screams at times, and mostly when I am in the room, so that it may be attributed to temper.

8 P.M., pulse 112, respiration 70, temperature 101.8°. Has slept a greater part of the day; would wake up and scream, but as he would take food with avidity and then go to sleep, I was still forced to attribute these outbreaks to anger; but those about him insisted that something else was the matter. The pulse was regular (see charts showing pulse, respiration, and temperature for the entire period), full and with very good tension—better than at any time during his illness. Respiration was easy, no evidence of any obstruction in either the upper or lower air passages, but very rapid; nostrils not dilated; no râles of any kind; no cough; skin cool and moist; vomited at 4 P.M., but two doses of the creasote mixture quieted the stomach; four greenish, watery stools. Had I compared the record of to-day with that of yesterday the prognosis would have been favorable; however, the enlarged cervical glands, which now obliterated the depression between the inferior maxilla and clavicle, the rapid breathing, and the drowsiness led me to anticipate trouble. So without alarming the sick, I instructed the nurse to send for me if she noticed any change for the worse. Nor was I mistaken, for at 1.30 A.M. (4th) they sent for me. I hastened to the house and on entering the room perceived a material change for the worse. He had brightened a little about 9 o'clock, had taken milk and beef-tea, and had fallen asleep. Between 10 and 11 he began to toss about; would give the most heartrending screams, point about the room as if he wanted something, but after trying to satisfy him in every way, and failing, they sent for me.

I found him tossing and screaming, pulseless, blue lips, countenance livid, respirations above a hundred and superficial, semi-conscious, face pinched, nostrils dilated, lungs œdematous and rapidly filling, surface bathed in a clammy sweat, and nose, hands and feet cold. Could the little fellow have now described his suffering, it would have vividly re-called the last moments of one of our late beloved members who said that he felt "as if bands of iron were constricting his chest." In less than half an hour, after a hard-fought battle, he died.

As I could not see that anything would be learned by a *post-mortem* examination it was not requested.

At the risk of being tedious I have minutely described the progress of the case, with the treatment. By doing this the therapeusis will be shown.

It is not my purpose to enter upon the subject of diphtheria, but rather to point out what I believe to be the interesting features of this particular case.

1. A point of great interest is the simultaneous development of the disease in both mother and child. They had been sleeping in the same bed for weeks, had enjoyed exceptionally good health, and at the same time, on the morning of Dec.

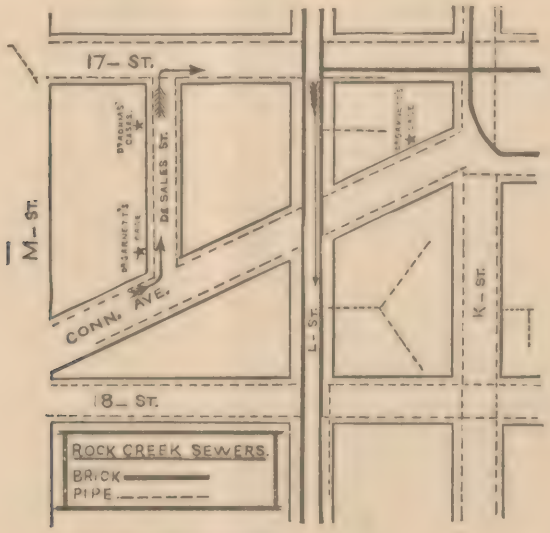
23d, though going about as usual, both manifested what turned out to be the initial symptoms. Because the membrane was recognized in the mother first, we are not justified in concluding that she infected the child, for the premonitory symptoms were exhibited in both about the same time, and, further, the alarming symptoms were first seen in the child. So I am forced to conclude that both were infected by the same contagium at the same time, and ran their courses simultaneously.

2. Whence came the poison? Here the cases are more puzzling than ever. The child had not been near any children outside of the house. The washerwoman had no one sick in her house, and had not washed for any sick person. The mother had been shopping on the 22d, but it is not reasonable to suppose that she contracted the disease in that manner. I was a visitor at the house, but as I had not seen a case of diphtheria for more than a year, the conveyance of the poison could not be traced to me. Is it reasonable to suppose that the father, who had been in all the large cities of the east, and who arrived on the night of the 22d, and slept in the same bed, was the medium of contagium? This can hardly be presumed, for he had neither seen nor heard of a case in his travels and never manifested the slightest symptom of the disease, though almost continuously in the sick-room.

By examining the accompanying plan of the north-west section of this city, it will be seen that the house of my patients is drained by a sewer begining at 17th Street and Rhode Island Avenue, thence by Connecticut Avenue, to De Sales Street, to 17th Street, to the large brick L Street sewer, which in turn empties into Rock Creek. I learned from Dr. A. Y. P. Garnett that, five days before my patients were taken sick, he was called to attend a case of diphtheria in a house at the Connecticut Avenue end of De Sales St., which was drained by the same sewer as the house of my patients.

May it not be a reasonable presumption that the poison was conveyed along this sewer and backed into the house at the 17th St. end? After the outbreak, a sanitary inspector condemned the plumbing and pronounced the house full of sewer gas. The plumber who put in the new apparatus, under the directions of the health officer, represented that the drip pan of

the closet was caked with filth that was offensive enough to sicken any one. But as I do not favor the spontaneous generation of the diphtheritic poison from filth, but believe it a poison *sui generis* which, with filth as a *nidus*, is a rapidly propagated one; and as the days of Dec. 20th, 21st, 22d, and 23d, 1883, were cold enough to freeze Rock Creek and thus cause an overflow of gas, I strongly favor the transmission of the coatagium from Dr. Garnett's case, through the sewer, to my own patients who slept in a room adjoining the bath-room, the communicating door being almost always open, and were consequently breathing air impregnated with sewer gas.[1]



3. What was the cause of this sudden death? Was it due to loss of heart power? I think not, because the heart had been acting remarkably well for sixteen hours, and when I left him at 8 P.M., the pulse was 120 with excellent tension. Was it because the stimulants had been withheld and the heart wore itself out? If this were so why did not the pulse show the necessity for re-stimulation? From the history I am compelled to exclude paralysis of the heart as the cause.

Was it due to traumatic pneumonia? I hardly think this

[1] I am at present attending a fourth case in the same house, a child two and a half years old.

tenable, because there was no paralysis of the muscles of deglutition, and no history of a foreign body entering the larynx.

Then was it due to paralysis of the lungs? During the whole course of the disease there was nothing unusual about the pulse-temperature-respiration ratio until the 3d of January, when the pulse fell and the respiration rapidly rose. This break in the ratio made me apprehensive. What caused these rapid, shallow respirations? If the respiratory centre was irritated by the poisoned blood, why did the circulatory centre escape? As the glandular enlargement before mentioned increased, the respirations became quicker. My opinion is that paralysis of the lungs caused my patient's death—or in other words, paralysis of the vagus. Whether the cause lay in the respiratory centre, or along the tract where pressed upon by the infiltrated cervical glands, I am not prepared to say.

Treatment.

If there ever was a case systematically managed such was this. Those in charge were intelligent people and at once realized the importance of carrying out every detail. Twice a day, the directions were written out, giving the hour each medicine was

CHESTER A——

January 2d, 1884.

WHISKEY—40 DROPS.	~~2~~ ~~4~~ ~~6~~ ~~8~~ ~~10~~ ~~12~~ P.M. ~~2~~ ~~4~~ ~~6~~ { VOMITING / 8–10–12 } A.M.
SPRAY.	~~4~~ ~~8~~ ~~12~~ P.M.
SOL. OF QUININE.	~~2~~ ~~10~~ A.M. ~~8~~ { SLEEP / 6 } ~~9~~ P.M.

to be given; the hour was to be crossed when it was given. By this means we were able to tell at each visit, how much of each drug had been taken (see table). Again, when the drug was not given there was a reason for it, and the nurse recorded it. By reference to the clinical notes it will be seen that on this date at 7 P.M. the child vomited, and had an irritable stomach the rest of the night, so that he missed the

whiskey. Again at the time for his quinine he was asleep, and soon after he awakened nausea supervened.

As an antipyretic, sodium salicylate has in my hands, in treating children, outranked the quinia salts. In this case the fever promptly subsided under its use, but with the subsidence came a depression of the heart which required free stimulation. Later our attempts to lower the temperature with quinine failed. It is remarkable how much stimulants children with diphtheria can bear. This child took large quantities without apparent effect upon pulse or fontanelle. It may be interesting to note that in two weeks' time these three patients drank fourteen quarts of whiskey. The bronchitis responded promptly to the ammonium carbonate. It may be thought that the patient in the earlier stage was not sufficiently nourished; I grant this if we exclude the alcohol, which can hardly be fair, since it is the best of foods. Later on he surely had enough food.

Having learned that Dr. Armistead Peter, of this city, recently lost a case, I wrote to him and was favored with the following:

"About 6 P.M. September 10th, 1883, Frank W., aged four years, passed my office apparently well. Same evening, nine o'clock, I was summoned to see him; he had a slight fever. Visiting him the next morning I found him covered with a decided scarlatina rash, pulse and temperature good, throat presenting the usual redness of mild scarlatina. September 12th, a well-marked diphtheritic exudation covered right tonsil, quickly spreading over uvula. Appetite, pulse, and temperature good. Case progressing favorably, and on the morning of the 19th throat clean; glands on both sides of neck (which were enlarged) resumed their natural condition, and the slight nasal discharge had ceased. I encouraged the parents with every hope of a good recovery, at the same time warned them that the sequelæ of scarlet fever and diphtheria were often more fatal than the primary disease, especially telling them of sudden deaths from heart trouble, having met with two cases during the last few years. The child was nursed with the greatest care, never allowed to make any effort to help himself or leave his bed. September 19th, 4 P.M., six hours after my morning visit, I returned home and found an urgent message to visit my patient. On arriving at the house was told of his sudden death, and Dr. Kleinschmidt, who had been called to see him at 3 P.M., informed me that at that hour he was suffering from paresis of the heart, but recovered (during this recovery his mother told me the child conversed and seemed to be all right); in about thirty minutes another attack

followed, and death was quickly the result. Drs. Grafton Tyler and Louis Mackall also saw the child, but had left the house before I arrived." "I have no theory to advance as to the pathological condition that causes these sudden deaths, except that there must be some latent poison in the blood, peculiar to the disease, that acts directly on the nerve centres. I call your attention to the theory of Edward Woakes, of London, who claims that sudden death is caused by paralysis of the vaso-motor nutrient vessels of the vagi."

"During the nine days' sickness the pulse and temperature were good, never exceeding 100°. Dr. Kleinschmidt saw him on the 16th, pronounced his general condition excellent; the edges of the diphtheritic deposit were then becoming ragged, inflammation subsiding, and, as I said before, on the morning of the 19th all traces of this deposit had disappeared, the tonsils and uvula looking clean and healthy."

In Dr. Peter's case the cause of death was undoubtedly paralysis of the heart. Dr. Kleinschmidt, who was called in when the first alarming symptoms occurred, and remained with the patient until his death, informed me that when he arrived at the bedside the heart had stopped, that he stood the patient on his head and compressed the chest, when the heart very quickly resumed its functions, and the patient recovered as described by Dr. Peter. When the second attack of syncope occurred the same means were resorted to, but the heart made no effort to act.

As the report of neither of these gentlemen states whether there was any undue muscular exertion, or whether the patient assumed the erect posture, of course we cannot conjecture the immediate cause of this sudden and repeated heart failure.

Dr. Ferguson,[1] of Canada, reports three cases of sudden death after the diphtheritic symptoms had subsided.

CASE I.—A young man had made a good recovery, with a slight paralysis of the right arm. He was sitting on a log and threw a stone at a small bird, when he fell over and expired in a few moments.

He attributes death to paralysis of the heart, and accounts for the blood found at post-mortem in the right heart as being passive, due to inspiration. The left ventricle was empty, and must have stopped in systole.

CASE II.—Was that of a little boy, aged five. He was apparently doing well, and while sitting on the chamber he complained

[1] Medical News, Dec. 15th, 1883.

of being weak, was put to bed and died in a few moments. Blood was found in both sides of the heart.

CASE III.—A girl, aged thirteen years, had recovered sufficiently to be in the parlor. While there she was affected so much by the death of a sister that she was carried to her room and died shortly after.

In commenting upon the cause of death in these cases, Dr. Ferguson says that it was not due to the poison affecting the nerve centres, for two of his cases had fairly recovered. In the case of the little boy, the heart's failure might have been due to the poison. Nor can he see how Woakes' theory is tenable in his first and third cases. "It claims that sudden death is due to a vaso motor paralysis of the vagi, so that these nerves are gorged with blood, and the fibrillæ suggillated to such an extent that they are unable to conduct inhibitory impulses, and the heart speedily wears itself out, being exhausted by a succession of rapid beats."

We cannot deny that the heart may be affected by strong emotions. The nerves supplying the heart are undoubtedly weakened by the poison in the blood that nourishes them. In such a condition emotions may act so powerfully, through the mind, on the heart as to produce a sufficient derangement of its action to cause death. Dr. Ferguson believes that the lesion is central.

Dr. S. J. Radcliffe,[1] of this city, reports a case of diphtheria following scarlatina. In half an hour after the doctor left him, apparently doing well, he was informed by a messenger that the child "sat up in bed to be fed, and while in the act suddenly fell back and expired." He says: "I considerered the remote cause of death in this case to be asthenia, and its immediate cause heart failure—failure to carry on its intrinsic work in the erect position."

Dr. Radcliffe seems to have lost sight of the probable existence of paralysis of the muscles of deglutition, and of those which protect the larynx from the entrance of foreign bodies during deglutition, and thereby the entrance of food causing death by apnea. He gives us no indications of loss of heart power, for in his brief clinical history he refers neither to the arterial tension nor to the pulse rate.

Dr. R. G. Mauss furnished me the following: Tracheotomy had been performed on a white boy, aged six, on the 6th day of

[1] Medical News, Jan. 5th, 1884.

diphtheria; was doing well when seen by the attending physicians at 8 P.M. At 9.30 the same night he shivered slightly and within five or ten minutes expired. Tracheotomy tubes were carefully examined and no obstruction discovered.

This history is too incomplete to venture upon the probable cause of death.

In the *Medical News* of January 12th, 1884, is an editorial based upon the four cases above referred to, and from which I shall quote at some length. The editor thinks that these cases are more to be dreaded because they occur at the time of supposed convalescence, when the patient's friends are full of hope, and a recovery has been predicted by the physician. "Very often the end comes when the patient, raised to the erect posture, is in the act of taking food." In such cases, death is attributed to failure of the heart, "when apnea has been induced by closure of the glottis by food or drink." Donders showed that paralysis of the muscles of deglutition is not uncommon in simple cases. The food not being properly conveyed along the proper tract, inspiration may take place and the food be drawn into the larynx; further, incomplete closure of the glottis may also allow particles of food to enter the larynx, and thus set up a fatal pneumonia.

Paralysis of the respiratory muscles may also take place, causing rapid edema of the lungs and sudden death. "The respiratory muscles may suddenly fail and death ensue with apnea."

"It is quite foreign to our purpose to consider the various esions of the heart which may occur in the course of diphtheria. We are here concerned with those changes which are common causes of sudden death. These are chiefly two: myocarditis or parenchymatous degeneration of the muscular tissue, and paresis of the cardiac motor ganglia. To the changes of the former may be added the effects of migrating micrococci colonies, and the latter is a part of a general change in the ganglia of the sympathetic. Damaged in respect to one or both of these parts, the heart may still be capable of carrying on its work when the body is recumbent. The fatal paralysis occurs when a change to the upright position requires the organ to put forth more vigorous efforts. Either the ganglia cannot produce the needed force, or the muscles prove unequal to the sudden strain, or both fail simultaneously."

"Another mode of sudden death—cerebral—there is reason to believe, does happen more frequently than is now supposed. We refer to cerebral hemorrhage and the so called serous apoplexy, both accidents which may attend on albuminuria. In making this statement, we are not unmindful of the convulsive phenomena and the direct interference with the respiratory centre, which may be due to the cerebral changes consecutive to albuminuria."

It may be claimed by some that my case is not properly classed and that it only ran the usual course of the disease. All the cases referred to, except one of Ferguson's, died during the convalescence. I do not claim that my patient had convalesced, but that that period was at hand when the acute symptoms had subsided, the membrane had disappeared, the pulse and temperature excellent, and when there was every indication of a speedy recovery. If my patient died as other cases, how can we account for the excellent pulse during the twenty-four hours preceding death?

What is meant by sudden death? Are we to conclude that only such cases as have been reported, where the patients were up and about, and from undue excitement or exertion dropped dead, are the only typical cases of sudden death? Surely, no one would feel justified in maintaining such an opinion. If a case is progressing favorably, and there is every prospect of a speedy recovery, when, from some unexpected change, whether the cause be known or not, the patient in a few hours succumbs, that is just as much a sudden death as if he were apparently well and attending to his accustomed duties, and should without apparent cause expire.

"The end of life may still be said to occur suddenly, even when the death struggle has lasted several hours. What is essential in the conception of this mode of death is the unexpectedness of the event relatively to the previous condition of the individual. The symptoms of agony last from a few minutes to a few hours, consisting often only in insensibility, insomnia, convulsions, and difficult breathing. A rapid death is frequent in the earliest years of life. . . . It is once again as frequent in males as in females."

"The causes of death are numerous, but the following divisions may be recognized:

Cessation of the circulation, through various diseases of the heart and great vessels.

Cessation of the respiration, through the action of various causes (suffocation and strangulation, deficient nutrition, either produced by albumen or water).

Extreme or long-continued elevation or diminution of the temperature of the body, and simultaneous deficiency of regulating means.

Severe physical and chemical agencies (purulent infection).

Severe psychical impressions.

Those organs which are the instruments of the most important activities of life, and whose injury most quickly causes death, were called by the ancients *atria mortis;* they are the heart, lungs, and the brain, or, to be more exact, the medulla oblongata. Hence we have, especially for the laity, three different modes of death: 1st, death from the brain called apoplexy (as in concussion of the brain, large extravasations); 2d, death from the respiratory organs, by asphyxia, or, more properly, by suffocation (as by the breathing of irrespirable gases); 3d, death from the heart (as, for example, in rupture of the heart). These three modes of death seldom, and only in sudden death, occur in pure forms. Usually, especially in slowly produced death, they combine with each other in various ways; for example, interference with the circulation alters the composition of the blood, and this, in its turn, impedes respiration, and both together diminish the heart's activity. In individual cases, even when a well-conducted autopsy is made, it is often difficult to determine the exact way in which death has been brought about."

By a study of these general causes of death, and comparing them with the history of my own case, I am compelled to exclude from the probable cause the last four. I cannot believe that my case died from deficient nutrition, for emaciation had not taken place to any marked degree, and at the time of death there were no evidences of exhaustion sufficient to cause death. Owing to the age of the child, it was impossible to obtain any urine for chemical analysis, so that I do not know whether albuminuria existed or not. If there was an excessive amount of water in the system, there were no evidences of it, for the pulse was good, and there was neither circumscribed nor

general edema. It was not until the death struggle set in that edema pulmonum began.

The variation in temperature could not have produced death, for the daily excursion was sufficient to give the nervous system a chance to recuperate.

There was no physical agency to account for the death. If it was due to the chemical agency (purulent infection), it would seem from the age of the patient as though death should have occurred some days sooner, and then too that there would have been more evidences of the system being overwhelmed by the poison.

In one so young, psychical impressions cannot be considered a factor in the causation of death. In the young lady referred to by Ferguson, the fright and grief at the death of a sister produced psychical changes sufficient to cause death in a very few moments.

Then I think in my case that I am warranted in concluding that death was the result either of cessation of the circulation or of the respiration, and that, from the history of the case, the heart held out longer than the lungs.

J. Lewis Smith says that death may result from: 1st, diphtheritic blood-poisoning; 2d, septic blood-poisoning; 3d, obstructive laryngitis; 4th, uremia; 5th, sudden failure of heart's action; 6th, suddenly developed passive congestion and edema of the lungs probably due to feebleness of the heart's action, or to paralysis of the respiratory muscles. In support of this last cause, he cites an instance where edema pulmonum occurred a month after the disappearance of the faucial membrane. The patient revived, but the following day the edema recurred and the patient died.

In order to understand how death might be produced by the destruction of the function of either of these organs it is necessary for us to study the nerve supply of the heart and lungs in a physiological sense. For this purpose, through the valuable assistance of Dr. Louis Kolipinski, of the Children's Hospital, D. C., I am enabled to give the generally accepted views of physiologists upon the complex workings of the nerve supply of these important organs.[1]

The following are the nerves of the heart: The plexus

[1] Landois, Lehrbuch der Physiologie des Menschen.

cardiacus is composed of the following nerves: 1. Ramus cardiacus of the vagus trunk and the branch, of the same name, from the ramus externus of the superior laryngeal, of the inferior laryngeal, and sometimes also from the plexus pulmonalis of the vagus—more often on the left side.

2. Of the ramus cardiacus superior, medius, inferior, and minus of the third cervical and first dorsal ganglia of the sympathetic.

3. The inconstant branch, the ramus descendens hypoglossi, which is said to originate from the first cervical ganglion (Luschka). From this plexus pass out the deep and superficial nerves; the latter, as a rule, receive a ganglion at the division of the pulmonary artery under the arch of the aorta. We can distinguish as going out of this plexus: *a.* Plexus coronarius dexter et sinister (Scarpa) which contain the vaso-motor nerves (physiological knowledge of these is still wanting), and also descending fibres (sensory to the pericardium) (?).

b. Nerves lying in the grooves and in the substance of the heart, which are richly provided with the ganglia (Remak) which we recognize as the automatic motor centres of the heart. A ring of nerves rich in ganglia borders the edge of the interauricular septum; another is found in the auriculo-ventricular margin; where these meet they interchange fibres.

The ganglia are situated principally near the pericardium. In mammals, the two larger lie near the insertion of the superior cava. In birds, the larger mass, containing thousands of ganglia, lies at the posterior intersection of the sulcus longitudinalis and transversalis. From these masses fine nerve fibres (bearing on them ganglia) penetrate the muscles of the auricles and ventricles.

In the frog, near the vagus fibres in the walls of the sinus of the cava, lies a large ganglionic plexus (ganglion of Remak). From this ganglion the vagus fibres pass as the anterior and posterior nerves of the septum, and each of these has a second ganglion (ganglion of the ventricle: Bidder's), from which can be traced, for a short distance only, nerve-fibres, so that the greater part of the ventricle seems to be without nerves.

By further analyzing the complex functions of this nerve-supply, we find that there are accelerator, automatic excitor, inhibitor, and vaso-motor nerves with presiding centres.

The action of the accelerator centre can only be tested after division of the splanchnics. This centre is not tonically excited, because section of the nerve does not slow the heart's impulse, and destruction of the centre in the medulla is without effect.

The heart has within itself automatic excitor centres, probably in its ganglia. These centres are connected with each other. They are regulated by the chief centre, which is in the cord. By irritation, these centres are stimulated into spasmodic action. The dominant centre is in the auricle. By depressing this centre (opium topically), the impulse may be transferred to the ventricles, and contraction may start from them. The centres of the auricles are the more irritable. Reflex irritation from the interior of the heart (endocardium) acts most markedly and rapidly. Blood-supply is a necessary part of the acting heart. In removing the auricular sinus, the remainder of the heart does not contract, because the ganglion of Bidder has not in itself the power of exciting the contraction, and also because the section irritates the inhibitory (vagus) nerve. Some hold that, in the heart, there are three ganglia—Remak's and Bidder's excitors and Ludwig's inhibitor. The first and second together are stronger than the third, but the third is stronger than either of the others.

The centre of cardiac inhibition (vagus) is seated in the medulla oblongata. The fibres of the vagus, when moderately irritated, diminish the number of heart beats; strongly irritated arrest them. They are from the spinal accessory. This centre is acted on directly and reflexly.

Directly: 1, Sudden anemia of the medulla; 2, sudden venous hyperemia; 3, increased quantity of carbon dioxide in the blood; 4, during inspiration; 5, increased blood-pressure in the cerebral arteries. Reflexly: 1, Irritation of sensory nerves; irritation of cerebral end of the cut vagus on one side; 3, irritation (blow on the sensory nerves of abdomen).

It is generally acknowledged that the vaso-motor centre is situated in the medulla near the calamus scriptorius. Irritation of this centre produces constriction of the arteries and rise of blood-pressure. It is directly excited by: 1. The composition of the blood as regards its gases; an excess of carbon dioxide produces constriction of the blood-vessels and engorge-

ment of the veins and heart, whereby the rapidity of the flow is increased. The same effect is produced by anemia of the medulla, and also by stagnation of blood in venous hyperemia. Reflexly excited by: 1. Pressor nerves which increase the vaso-motor action, inferior and superior laryngeal, trigeminus, cervical sympathetic, and any sensory nerve of the body (Lovin).

2. Depressor nerves, which inhibit the action of the vaso-motor centre; depressor of Cyon; vagus. Also said to be found in all sensory nerves.

Paralysis of the vaso-motor nerves causes the heart to contract slowly and feebly, the reverse when the vaso-motors contract the blood-vessels. Paralysis of the splanchnics may so fill the abdominal vessels with blood that death may result from anemia of other organs, *i. e.*, intra-vascular death from loss of blood. The phrenic, spinal, spinal accessory, and vagus nerves are the principal ones concerned in the mechanism of respiration, and their distribution must be understood in order to intelligently study the action of the respiratory centre.

Le Gallois recognized that the respiratory centre must be contained in the medulla. Flourens determined more accurately its seat to be behind the origin of the vagus, on both sides of the calamus scriptorius of the fourth ventricle, and between the vagus and spinal accessory nuclei. He called it *Point, or Nœud, Vital.* This centre is bilateral, and can be separated by longitudinal section (Longet, Volkmann, Schiff), and thereby respiration is performed symmetrically on both sides. When a vagus trunk is cut, respiration is slowed on that side; on double section bilateral respiration is the same. Irritation of the cerebral end of a cut vagus stops the respiration on that side; the same result is attained on irritating the trigeminus (Longendorff). On transverse section of one centre, respiration stops on that side. This centre is composed of two alternately acting centres, one inspiratory, the other expiratory. It is automatic. Its excitability depends on the quantity of oxygen and carbon dioxide in the blood; hence we have apnea, empnea, dyspnea, and asphyxia.

Dyspnea may be caused by: 1. Any agent preventing the action of the respiratory organs, as stenosis of the air-passages, pneumonia, etc. 2. Prostration of the circulation, as

heart disease, obstruction to the flow of blood from the brain, etc. 3. Hemorrhage.

Irritation of the peripheral end of the cut vagus stops the heart and also the respiration for a short time, because of anemia of the medulla.

The centre is excited reflexly by the pulmonary branches of the vagus, the nerves of the eye, ear, and skin.

It is reflexly depressed by the superior and inferior laryngeals, nasal branches of the trigeminus, sensory nerves of the skin, of the thorax and abdomen, and by pressure on the brain.

It must be recognized that, if paralysis of either the cardiac, respiratory, or vaso-motor centres takes place, death quickly follows. These three centres are situated in the medulla oblongata. Their powers of resistance must be different, for only one seems to be affected at a time. Whether this sudden overpowering of the centre which causes the sudden dissolution is due to the millions of micrococci, to a specific unknown poison, or to an individual idiosyncrasy incapable of combating with the ordinary diphtheritic poison, remains to be discovered.

Let us suppose a case of sudden death from paralysis of the heart. According to Dr. J. Lewis Smith, it must be due to one of three causes—anemia, granulo-fatty degeneration of the muscular fibres of the heart, or to ante mortem blood clot. The first is the potent cause which, by enfeebling the heart, causes less oxygenated blood to circulate through the respiratory centre, which is paralyzed, and death results from asphyxia. The respiratory centre is composed of the nuclei of the vagus, trigeminus, spinal accessory, and glosso-pharyngeus (Meynert), and hence asphyxia means paralysis of these nerves. But suppose it be due to the degeneration of the muscular fibres. We are well acquainted with the fact that the muscular fibres of the heart are different from those of the general muscular system, for, by irritating the sympathetic, it is impossible to produce a tetanic contraction of the heart's muscles, when by a similar irritation this contraction can be produced in muscles generally. Further, by irritating the pneumogastric, we are not able to stop a contraction of the muscles already begun, but merely to prevent the succeeding beat. If, then, from any cause, this peculiar muscular fibre be destroyed,

the central mass must lose its influence, and the cause of death will certainly be failure of the heart.

Or let it be due to ante-mortem blood-clot. The pneumogastric nerve might be irritated by food taken into the stomach, and the heart stopped just long enough to form a clot. Energetic treatment may overcome this attack of syncope (as in Dr. Peter's case), but a second attack probably carries off our patient by pulmonary embolism.

But if it be due to passive congestion and edema of the lungs from feebleness of the heart's action, the same central mass is affected, the primary cause being from without. In this case the heart stops after the respiration. Asphyxia irritates the inhibitory nerve, thereby slowing the heart's action, but stimulates the vaso-motors, whereby the venous system and heart are full of blood, and arterial tension is increased.

This destructive element, whatever it is, may be circulating in the blood and be continually irritating the cardiac centre. This central mass may, through its power of resistance, protect itself for a long time, but constant irritation must necessarily reduce the resisting power, and paralysis is the result.

If this destructive force is concentrated on the inhibitory centre, what will be the result? This nucleus being the cardiac moderator, if its power is lost, the heart will soon wear itself out by a succession of rapid pulsations. But the influence of the vagus on the respiratory movements must not be lost sight of; paralyze it, and the respirations become slower and slower, until they finally stop in expiration.

The automatic centres may be the ones seriously affected. They are governed by a chief centre in the cord, whose power of resistance ought to be equal to that of the central mass.

Destruction of the accelerator centre seems to be without effect, so its paralysis cannot be considered a prime factor in the causation of sudden deaths.

Let the vaso-motor centre be the paralyzed element, and what is the result? Tht heart contracts slowly and feebly, arterial tension is diminished, and the venous system is engorged, and death may result from congestion of the internal organs.

But let this destructive element expend its force on the respiratory centre. The effect will most likely be paresis of the

diaphragm and lungs, edema of the lungs rapidly supervening. This infiltration causes rapid superficial respirations (dyspnea), the blood is not sufficiently oxygenated, and, surcharged with carbon dioxide, is conveyed to the central masses. Hence the respiratory centre, being constantly irritated by this non-oxygenated blood, calls on the auxiliary centres presiding over the muscles of forced respiration, which for a short time supply the place caused by the paralysis of the phrenic and vagus. But as the quantity of carbon dioxide increases, the vaso-motor centre is stimulated, the heart beats become slower and stronger, the vascular tension increases, and thus nature endeavors to compensate for the impairment of the lungs.